

## A brief review of infectious and parasitic diseases of wapiti, with emphasis on western Canada and the northwestern United States

Judit E.G. Smits

### Abstract

In this paper I review diseases reported in both captive and free-ranging wapiti in western North America, with some reference to diseases in captive red deer in Great Britain, Europe, New Zealand, and eastern North America.

With the exception of coronavirus in neonates, few viral agents are reported to cause serious disease losses in wapiti in North America at this time. Bacterial diseases of current significance include brucellosis (focus in Wyoming), clostridial diseases, coliform enteritis of neonates, pasteurellosis, and necrobacillosis. The endoparasites most likely to be seen causing lesions in wapiti of western North America are lungworm (*Dictyocaulus viviparus*), arterial worm (*Elaeophora schneideri*), and, possibly, liver fluke (*Fascioloides magna*). Ectoparasites of importance to wapiti are *Psoroptes cervinus* and *Dermacentor albipictus*. Nutritional diseases are not covered in this review.

### Résumé

Revue succincte des maladies infectieuses et parasitaires chez le Wapiti dans les régions de l'Ouest canadien et du Nord-Ouest des États-Unis

L'auteur procède à la revue des maladies rapportées chez le Wapiti en liberté et en captivité dans l'ouest de l'Amérique du Nord et inclut certaines références concernant les maladies affectant le Cerf de Virginie en captivité en Grande-Bretagne, en Europe, en Nouvelle-Zélande et dans l'est de l'Amérique du Nord.

À l'exception du virus corona chez les nouveau-nés, il y a peu d'agents viraux identifiés comme agent causal de maladies entraînant une perte sérieuse chez le Wapiti en Amérique du Nord. Les infections bactériennes significatives principales incluent la brucellose (un foyer au Wyoming), les maladies à clostridium, les entérites à *E. coli* chez les nouveau-nés, la pasteurellose et la nécrobacillose. Les endoparasites les plus susceptibles de causer des lésions chez le Wapiti de l'ouest

de l'Amérique du Nord sont les vers du poumon (*Dictyocaulus viviparus*), le vers des artères (*Elaeophora schneideri*) et possiblement la douve du foie (*Fascioloides magna*). Les ectoparasites ayant une importance chez le Wapiti comprennent *Psoroptes cervinus* et *Dermacentor albipictus*. Dans cette revue, l'auteur n'a pas inclu les maladies d'origine nutritionnelle.

(Traduit par Dr Thérèse Lanthier)

Can Vet J 1991; 32: 471-479

### Introduction

There is a current and growing interest in the raising of wapiti (elk, *Cervus elaphus canadensis*) in captivity for venison production. Game farming is a new industry that is developing in response to global demands for low cholesterol, high quality meat. Wapiti are especially popular for farming in western Canada because they are a gregarious, native species, well adapted to the varied conditions and climate of the western Great Plains and Rocky Mountains. Presently, there are approximately 8,000 farmed wapiti in western Canada, and 240 game farmers in western Canadian game farmers' associations.

The main purpose of this review is to provide some information on diseases of wapiti for veterinarians who find themselves working with this species. Published information is not readily accessible to most practitioners.

In this paper I review the literature on diseases and conditions of wapiti, with emphasis on western Canada and the northwestern United States. The review was accomplished through use of the annotated bibliography, "Parasites and Diseases of North American Elk (*Cervus* spp.)" issued by the Montana Department of Fish and Game in cooperation with the Montana Agricultural Experiment Station, Montana State University, Bozeman, USA in 1976. Data bases that were searched for relevant information from 1976 onward were BIOSIS (Biological Abstracts Inc.) and CABI (Commonwealth Agricultural Bureau International). Key words used were: disease, bacteria, viruses, parasites, elk, wapiti, cervidae, and deer. Other sources included proceedings from deer production symposia

Department of Herd Medicine and Theriogenology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0.

This work was supported in part by a federal ERDA grant.

from New Zealand, Australia, and Great Britain. The inclusion of references to diseases in red deer (*Cervus elaphus*), wapiti, and wapiti x red deer hybrids in New Zealand and Great Britain was felt to be relevant. Red deer are conspecific with wapiti and thus may provide insight into the potential susceptibility of wapiti to certain diseases.

Bacterial and viral diseases of biological or economic significance in the captive and free-ranging wapiti populations are covered first. Parasitic diseases are then discussed, followed by a short discussion on plant toxicoses.

## Bacterial diseases

*Actinomyces* (*Corynebacterium*) *pyogenes* has been isolated from a wide variety of lesions in wapiti, including omphalitis and visceral abscessation in neonates, and purulent bronchopneumonia, footrot, arthritis, osteomyelitis, and abscesses in older animals (1). *Actinomyces pyogenes*, a ubiquitous organism, may require predisposing conditions to cause the lesions described.

Brucellosis is a disease of national and international concern, and exists as a localized problem in the northwestern United States. It is caused by *Brucella abortus* and is highly contagious. Outside of enzootic foci, it usually occurs as sporadic cases in wapiti that have been infected by primary host species such as cattle (2). Infection is through oral contact with aborted fetuses, and licking or ingestion of fetal fluids and vaginal exudate. Clinical manifestations include abortion, the birth of stillborn and nonviable calves, hygromata, and synovitis (3). There is persistent *B. abortus* infection in the wapiti herds of Wyoming's National Elk Refuge and Yellowstone National Park in the United States, where studies have demonstrated up to 50% prevalence of infection (2). In the problem area, park management includes winter feeding programs, and abortions occur at these feeding stations. Control of this focus of infection would most likely require cessation of feeding programs in order to stop the congregation of infected animals which perpetuates the infection. A measure being taken in an attempt to increase calf populations, and to decrease the potential of transmission of the disease to livestock, is the Stain 19 Brucella vaccination program presently underway in these populations (E.T. Thorne, personal communication).

Detection of brucellosis in ruminants is with the buffered brucella antigen (BBA) or rapid card test conducted on plasma or serum. This test may be supplemented with other seroagglutination tests, or complement fixation tests (2).

Brucellosis has not been detected in other wapiti in the United States or Canada.

All ruminants show various degrees of susceptibility to infection by clostridial organisms. Confirmed reports of clostridial disease in wapiti are not common. *Clostridium perfringens* has been isolated from captive wapiti calves (1) and red deer (4) with enterocolitis and enterotoxemia. Enterotoxemia in deer, caused primarily by *C. perfringens* type D, can be precipitated by a sudden change from a low to a high energy ration (5). Diagnosis is often speculative, based upon findings such as rapid postmortem decomposition plus

a history of dietary change that would be consistent with the development of enterotoxemia (4,5).

Blackleg and malignant edema are other diseases seen in wapiti. They may result from anaerobic conditions allowing the sporulation of clostridial bacilli in superficial wounds or in deeply damaged muscle. In young wapiti, such infections with lesions of gas gangrene, subcutaneous and intramuscular hemorrhage, edema and emphysema of the extremities, and serosanguineous thoracic and abdominal exudate, have been described (6). Species of *Clostridia* that have been involved in these cases include *C. chauvoei*, *septicum*, *sordelli*, and *perfringens* (5,6).

The most susceptible age for either of these types of clostridial disease among ungulates is thought to be between six months and two years. Routine annual prophylactic vaccination of captive wapiti with a multivalent clostridial vaccine is recommended (4). Work with red deer in New Zealand has not demonstrated sustained antibody titers three months after vaccination (7). Therefore, the timing of vaccination is recommended to be immediately prior to the period of maximum vulnerability: males prerut, females before calving, and calves at weaning.

Diarrhea and enteritis due to *Escherichia coli*, with or without septicemia, is a major cause of mortality among neonatal wapiti (1), as it is with neonates of most domestic species. *Escherichia coli* has also been isolated from cases of meningitis, footrot, and nephritis (1). Bacterial culture of affected tissues can provide a definitive diagnosis.

Predisposing conditions for the development of colibacillosis include stressors such as unsuitable environments, hand rearing, or infection with viral or parasitic agents which potentiate the invasion and systemic spread of gastrointestinal coliform bacteria (8,9). Control of diseases associated with *E. coli* would entail environmental amelioration.

All ruminants are probably susceptible to *Mycobacterium paratuberculosis* (*M. johnei*) infection. Paratuberculosis, or Johne's disease, has been reported in free-ranging and captive cervids in North America, Great Britain, Europe, and New Zealand (10-14). Paratuberculosis is a chronic wasting disease in most ruminants. In deer it tends to be more acute and has been observed to primarily affect deer between one and two years of age (12-14). Clinical signs associated with natural and experimental infection with *M. paratuberculosis* in elk, red deer, mule deer, and white-tailed deer are nonspecific. Failure to gain weight, and poor shedding of hair coat, are commonly observed. In natural infections reported in red deer, rapid weight loss and diarrhea do not occur until shortly before death (12,13). Diarrhea is not a consistent finding, although it was reported in tule elk with confirmed paratuberculosis (14).

The current status of free-ranging and captive wapiti populations with respect to Johne's disease is unknown. However, with the social stressor associated with increased population densities and captive environments, and the knowledge that "outbreaks" of Johne's disease have been described in red deer (12,13), it must be considered as a potential problem for captive wapiti in North America.

Antemortem diagnosis is most reliably based upon a combination of history, clinical signs, and the demonstration of acid-fast organisms in fecal smears, or the culture of *M. paratuberculosis* from feces (12-14). A new DNA test has been developed to confirm Johne's disease in clinical and preclinical cases, but its sensitivity and specificity for cervid species are not yet established (15).

Effective control of this disease has not been reported. However, British workers feel that vaccination with *M. paratuberculosis* in oil adjuvant can control clinical paratuberculosis despite its failure to eliminate infection (13).

Infection with *Fusobacterium necrophorum*, which causes local or systemic necrobacillosis, is a very important problem in wapiti (16,17). Necrobacillosis can be expressed as any combination of laryngitis, stomatitis, reticulorumenitis, and footrot. There have been epizootics of necrobacillosis on winter feeding grounds of wapiti in Wyoming, where 10% of the herd died each winter for fourteen years consecutively (16).

In order to gain entry and establish infection, the organism requires previous damage to the skin, hooves, or mucous membranes. Wet or contaminated conditions may lead to infection of the foot, coarse feed may contribute to stomatitis, and very fine feed may lead to ulcerative rumenitis (17).

High mortality rates, seen primarily in young animals, are caused by embolic spread of the infection from the primary site(s) to pulmonary or hepatic tissue. Death is usually due to ensuing pneumonia or toxemia. Adults tend to develop chronic infections from which they can recover (17).

Outbreaks have been associated with overcrowded, muddy conditions, so disease control would include the improvement of predisposing environmental conditions.

Suppurative bronchopneumonia associated with *Pasteurella multocida* causes mortality in adult wapiti (1,18). Fatal septicemic pasteurellosis, an uncommon disease in North America, was seen in 38 wapiti on the National Elk Refuge in Wyoming (18). Mortality was preceded by a short course of severe depression in the affected bulls, cows, and calves. Diagnosis was confirmed by isolation of *P. multocida* from affected tissues.

Septicemic pasteurellosis, endemic in various species in the tropics, is known to occur more frequently during the rainy season in which temperatures fluctuate. The outbreak on the National Elk Refuge also corresponded to a period of extreme weather fluctuation (18).

*Pasteurella multocida* is a normal inhabitant of the upper respiratory tract of wapiti, and may become pathogenic under conditions of nutritional, social, or environmental stress (17,18).

Tuberculosis (TB), a contagious zoonotic disease caused by *Mycobacterium bovis*, has worldwide distribution. In the early 1980s there was an outbreak of TB in several confined wapiti herds in North and South Dakota (1,19). Clinical signs were nonspecific. Affected animals were in poor condition, showed decreased exercise tolerance, and separated themselves from the herd. Some wapiti had chronic subcutaneous

abscesses which did not resolve with treatment. Necropsies of all the clinically affected animals revealed granulomatous pulmonary lesions suggestive of TB, although only two of 10 cases were confirmed as *M. bovis* on culture (19).

Because TB is of public health concern, and transmissible among numerous species besides humans and ungulates, all game animals imported into Canada must now undergo a series of cervical intradermal tests before they are released from quarantine. The caudal fold test is considered unreliable and is not acceptable in cervid species (20).

Reports of TB in cervids in North America have been rare (19). However, in situations of intensive management, or in the presence of sylvatic hosts or infected bovids, this disease could become a serious problem, as is the situation in farmed deer in Great Britain and New Zealand (21,22).

Yersiniosis, caused by *Yersinia pseudotuberculosis*, causes disease in numerous mammalian and avian species (23). Clinical disease has not been reported in wapiti in North America. Ten positive *Y. pseudotuberculosis* fecal cultures from deer (species unspecified) were discussed in a report on the incidence of pseudotuberculosis infections in Canada over a 23 year period (23). Yersiniosis is one of the leading causes of mortality in young farmed deer in Great Britain (5) and New Zealand (24,25). Frequently seen as sudden death, it may or may not be preceded by dysentery and depression. In endemic areas, *Y. pseudotuberculosis* is found in the gastrointestinal tract of healthy as well as diseased animals. Therefore, diagnosis depends upon clinical signs and necropsy findings, as well as isolation of the organism (24,25).

*Yersinia enterocolitica*, distinguishable from *Y. pseudotuberculosis* by biochemical tests (26), is known to cause enteric disease in numerous mammalian species (26,27). *Yersinia enterocolitica* has been recovered from 13 to 38% of fecal samples from wapiti surveyed in northwestern California, but no clinical cases of yersiniosis have been seen (27).

Reports of yersiniosis in muskoxen of northern Canada in 1986 indicate the presence of pathogenic yersinia in Canada (28), although it has not been seen in the southern regions yet. The hemorrhagic enteritis seen in muskoxen is similar to the disease in red deer in Scotland (5) and New Zealand (25), suggesting that yersiniosis could become a problem in free-ranging or captive wapiti.

Development of fatal yersiniosis is associated with environmental stressors such as cold wet weather (5,25), so environmental improvement would help control disease outbreaks.

Infection with the rickettsia *Anaplasma marginale* in wild ruminants is seldom associated with clinical disease (29,30). Subclinical anaplasmosis and inapparent infection detected by seropositivity have been reported in wapiti in the northwestern United States (29,30). Experimentally infected wapiti have shown mild, transient anemia followed by recovery with persistent inapparent infection (30).

Detection of seropositivity in wapiti is important for import requirements, and for the protection of cattle which are susceptible to anaplasmosis. Serum is tested

by the Rapid Card Agglutination (RCA) test or the Modified RCA in the United States (29,30), and by the complement fixation in Canada.

*Listeria monocytogenes* has been isolated from mammals, birds, aquatic animals, soil, silage, and water (31). *Listeria monocytogenes* has been incriminated in cases of late gestational abortion and in the birth of weak, nonviable calves in farmed red deer in Great Britain (32). A survey for the presence of this organism in feces of wild wapiti populations in the western United States showed a higher isolation rate among wapiti frequenting aquatic habitats, but there was no evidence of a detrimental effect on that population (31).

Leptospirosis is an infectious, zoonotic disease that affects many species of livestock and wildlife. It has not been implicated in clinical disease in wapiti, although a survey in Alberta showed 25% seropositivity among 28 wapiti tested. Serovars that were detected included *Leptospira autumnalis*, *L. bratislava*, and *L. icterohemorrhagiae*. Only 6 of 117 cattle tested had serovars matching those found in the wapiti from the same regions (33). In another study, serological samples for leptospiral serovars *pomona*, *hardjo*, *icterohemorrhagiae*, *canicola*, and *grippotyphosa*, from approximately 2,000 wapiti in Colorado, all tested negative for leptospirosis (34).

## Viral diseases

Bluetongue (BT) virus and epizootic hemorrhagic disease (EHD) virus are closely related orbiviruses. A Canadian outbreak of EHD and BT in 1987 involved wapiti, cattle, mountain sheep and goats, white-tailed deer, and bison (35).

In clinical trials involving infection of wapiti with BT virus (36) and EHD virus (37), BT caused mild pyrexia, conjunctivitis, and diarrhea on days 9 and 10 postinoculation, while EHD produced only mild fever. Wapiti infected experimentally with BT virus developed viremias sufficient to infect the major North American vector, *Culicoides variipennis* (36,37). Experimental infection with EHD also produced a viremia up to 30 days postexposure. Because of this, there is some concern that wapiti may be long-term carriers of these viruses and thus could serve as reservoirs of infection.

Agar gel immunodiffusion remains the screening test of choice for orbiviruses. Serum neutralization tests then are used to differentiate the various serotypes of EHD and BT (38). There is constant surveillance and serological testing of wapiti and other livestock entering Canada since BT is federally designated as a "reportable disease".

Contagious ecthyma (CE), also known as Orf or Soremouth, is caused by a parapox virus. This virus affects wild and domestic sheep and goats, reindeer, antelope, and muskoxen (39) as well as red deer and red deer x wapiti hybrids in New Zealand (40). The disease has been confirmed by electron microscopy (EM) in a wapiti, according to pathology records at the Western College of Veterinary Medicine.

Infected animals temporarily develop proliferative vesicles, pustules, and crusty lesions primarily on the lips and muzzle (39), but lesions have been seen on the

neck, legs, thorax, and perineal regions (40). An epizootic of contagious ecthyma in farmed red deer in New Zealand occurred between November 1985 and March 1989. Morbidity rates in some herds were up to 100%, and mortality associated with CE was seen on one farm (40).

Diagnosis is based upon clinical signs, with histopathological or electron microscopic confirmation.

The virus can persist for long periods in the environment in scab material. This virus is potentially zoonotic, and humans may develop vesicular lesions from careless handling of clinical cases of CE.

Coronaviral infection has been identified as a cause of neonatal enteritis in wapiti. In one study, this virus was the only pathogenic organism isolated from five of 11 calves with enteritis, some of which had concurrent colitis and abomasitis (1). Control of infection would entail minimizing contamination and stress in the environment of the neonate.

A syndrome described as chronic wasting disease (CWD) in wapiti at wildlife research facilities in Wyoming and Colorado has been described in mule deer, black-tailed deer, and mule deer x white-tailed deer hybrids previously inhabiting the same facilities (41). Behavioral change was accompanied by progressive, severe weight loss. The apparently long incubation period of three to five years, and the spongiform encephalopathy seen in all affected animals, are very similar to scrapie in sheep and goats. Diagnosis of this disease is based upon the clinical course of the disease plus CNS lesions. Unlike scrapie, vertical transmission is not thought to occur in similar diseases in other species, including CWD in cervids, bovine spongiform encephalopathy (BSE) in cattle, and kuru and Creutzfeldt-Jakob disease of humans (42). Contact with infected animals appears to play a role, but little else is known about transmission of CWD.

Herpesvirus of cervidae type 1 (HVC-1) has been isolated recently from farmed and free-ranging red deer in Great Britain and Belgium. Neutralizing antibodies of HVC-1 were detected in 29% of 520 farmed or free-ranging red deer tested in Great Britain (43), with a much lower prevalence among red deer in Belgium (6 of 20), and in deer in France (1 of 80) (44). Whether HVC-1 occurs in North American wapiti is not known, although 10 of 22 wapiti in a survey in Alberta showed antibody titers to bovine herpesvirus 1 (IBR) (33), which is antigenically related to HVC-1 (43).

Clinically, the virus is associated with conjunctivitis, ocular discharge, photophobia, and edema of the eyelids (44). A few cases have resulted in corneal rupture (43). Outbreaks involve mainly weaned calves. Diagnosis is based upon serum neutralization titers from acute and convalescent serum samples of clinically affected animals. Viruses may also be isolated from ocular and nasal swabs and identified by EM. The significance of HVC-1 in North America is not yet known, but it should be considered in unexplained cases of ocular lesions in wapiti.

The etiological agent of sheep-associated malignant catarrhal fever (SA-MCF), a disease of ungulates that are associated with sheep at lambing time (45,46), is unidentified. Wildebeest-associated MCF (WA-MCF)

is caused by alcelaphine herpesvirus 1. Although MCF affects ruminants worldwide (45–49), the susceptibility of wapiti is unknown.

Both forms of MCF cause the same clinicopathological syndrome affecting the gastrointestinal and upper respiratory tracts, eyes, and lymphoid organs (47). Clinical cases are almost invariably fatal.

In New Zealand, where MCF is one of the major diseases of farmed deer, wapiti x red deer hybrids appear to be more resistant than red deer, which are more resistant than Pere David's deer (48). Malignant catarrhal fever remains a potential threat to captive wapiti populations. Avoiding contact between deer and sheep may help control MCF in deer.

Rotavirus was the sole organism isolated from captive wapiti calves in western Canada and the United States which died with clinical signs of pneumonia and diarrhea (1), suggesting a causal relationship. The importance and prevalence of this viral infection in captive and free-ranging wapiti calves has not been studied.

Vesicular stomatitis (VS), caused by a rhabdovirus, produces periodic epizootics in livestock. There is serological evidence of infection by VS virus in enzootic foci in mule deer, wapiti, and pronghorn antelope populations (50). Serological surveys of local free-ranging populations showed neutralizing antibodies to VS virus in 4% of wapiti before, and 10% after (mule deer 4% before, and 20% after), the 1981 epizootic of VS in the Rocky Mountain states (50). These findings indicate definite exposure of wapiti to VS, but the clinical significance, if any, is not clear.

Rabies, also caused by a rhabdovirus, has been reported in red deer in Europe (51) and in a wapiti at a quarantine facility in Ontario (M. Bringans, personal communication). Rabies must be considered as a differential diagnosis if central nervous system signs are evident. Diagnosis is based on fluorescent antibody tests and mouse inoculation with suspect brain tissue, or, most recently, neuroblastoma tissue culture techniques. Histopathological lesions are not reliable criteria since inclusions indistinguishable from negri bodies have been detected in the brains of normal red deer and moose (*Alces alces*) (52,53).

## Parasitic diseases

The impact of gastrointestinal nematodes on free-ranging and captive wapiti populations is not clearly established. In spite of numerous studies on prevalence and types of parasites found in wapiti (54–56), there is very little information from controlled studies on the effects of intestinal parasitism. Studies on farmed red deer in New Zealand (57) and wapiti in North America (54) did not consider correlations between fecal egg counts, associated worm burdens, and the clinical significance of these burdens. In spite of high stocking densities on some deer farms and parks, there are few cases reported of uncomplicated gastrointestinal parasitism. Bison from similar environments do tend to suffer from health-threatening parasitic burdens (58).

This paucity of information on the pathological significance of gastrointestinal nematodes in wapiti suggests that these worms are not associated with

serious clinical disease at this time. Perhaps some degree of host-parasite balance has evolved with the cervid species.

## Lungworms

*Dictyocaulus viviparus* is common in wapiti and red deer. It is felt to be the most important parasite of wapiti in the northern Rocky Mountain region of Canada and the United States, as well as in New Zealand and Great Britain (54,56,57,59,60). Wapiti of all ages are affected. Ruminants acquire infestation by ingestion of third-stage larvae while feeding. The highest frequency of infection with *D. viviparus* occurs during spring "green-up", and it decreases through the fall and winter (56,59,60). Nutritional status and physiological condition appear to be the most significant factors in the wapiti's ability to deal with lungworm burdens. There is no evidence of acquired immunity to reinfection (54).

According to a New Zealand study (57), the intensity of infection with *D. viviparus* in red deer is highest in those under one year of age.

Experimental infection of bovine calves with *D. viviparus* larvae from Rocky Mountain elk failed to produce patent infection or clinical signs of infection (61) nor did wapiti calves inoculated with bovine strains of this lungworm develop signs of infection (61). A Scottish study showed *D. viviparus* of bovine origin to be readily transmitted to young red deer, but infection failed to produce clinical disease in well-nourished red deer calves (62). Variable results are presented on the cross infectivity of this lungworm between cervid species and cattle, but, in general, evidence suggests that cross infection between cervid species and cattle is not a problem (61,62).

## Meningeal worm

The protostrongylid worm *Parelaphostrongylus tenuis* occurs in most parts of eastern North America, from western Manitoba south to eastern Oklahoma and Texas and east to the Atlantic coast (63–67). Meningeal worm infections in cervids are acquired by the ingestion of infected gastropods. In the natural white-tailed deer host, the adult worms are associated with the subdural space and cranial venous sinuses. In nonhost cervids, including wapiti, infection with *P. tenuis* can cause moderate to fatal neurological disease due to aberrant behavior and migration of the worm, and increased host susceptibility (63–66,68).

There have been pathological findings consistent with meningeal worm infection in free-ranging wapiti sharing range with white-tailed deer. These include neurological signs, the recovery of female nematodes from the central nervous system (CNS), dorsal spined larvae in the feces, and the identification of typical histological lesions in the CNS (63–66). *Parelaphostrongylus tenuis* can only be positively identified on morphological characteristics of adult male worms (66).

At this time, meningeal worm has not been found in Canada west of the Manitoba-Saskatchewan border (67). There is considerable concern about the spread of this parasite and its serious potential impact on susceptible cervid populations.

There is some evidence that wapiti can survive infection and shed larvae in their feces (66–69).

#### Tissue worm

*Elaphostrongylus cervi*, another protostrongylid worm, is also called the tissue worm. Wapiti become infected by ingesting snails or slugs containing third-stage larvae. This worm invades the thoracic musculature and/or CNS or affected cervids. It has been known to cause neurological disease in red deer and wapiti in New Zealand (70,71), red deer in Scotland and Hungary (72,73), and *Rangifer* spp. in the USSR, Scandinavia, and Canada (the province of Newfoundland) (70). Because of the known susceptibility of wapiti (71) and other ungulates in North America, there are strict importation conditions placed on wapiti and red deer coming into Canada from infected areas.

#### Arterial worm

The arterial worm *Elaeophora schneideri* is a filarial nematode known to infect wapiti in western North America (54,74). Transmission of infective larvae depends upon contact among tabanid vectors, infected hosts, and susceptible wapiti or other ruminants.

The definitive host, mule deer, shows minimal or no clinical signs. Wapiti calves and yearlings in populations at elevations above 6,000 feet are most commonly affected (75). Infection has rarely been observed in wapiti of the northern Rockies (54).

The adult worm lives in the carotid artery or its branches; the occlusion of smaller arteries can cause ischemic necrosis of superficial parts of the head. Blindness, ear tip and muzzle necrosis, antler deformity, CNS necrosis, and renal and hind limb arteritis have been described in wapiti, an abnormal host (54,74,75).

#### Flukes

Wapiti are one of the normal hosts of the giant liver fluke *Fascioloides magna*, along with white-tailed deer, caribou, and possibly mule deer (76). As aquatic snails serve as intermediate hosts, the infection has been described most frequently in moist environments. It has been an infrequent problem in Saskatchewan (76). One survey in Alberta indicated that 9% of yearlings and 29% of adult wapiti are infected with *F. magna* (77). Surveys in southwestern Alberta (33) and in Banff and Kootenay National Parks (M.J. Pybus, personal communication) indicate a very high prevalence of the fluke in adult wapiti (45% and 86%, respectively). Studies during the 1950s and 1960s reported no infection, or low prevalence of infection among elk (56). These recent data suggest movement and establishment of the parasite into new areas (33,77).

Bison, cattle, and moose are aberrant hosts. They develop cysts which do not communicate with the biliary system. The ensuing hepatic lesions may be severe and are called "liver rot". In sheep the infection is frequently fatal (78). Although *F. magna* is thought to be nonpathogenic to the normal host, there is very little published concerning the clinical significance of this fluke in wapiti.

#### Tapeworm

Wapiti are intermediate hosts for cestodes of carnivores throughout regions of shared habitat. The

cestodes include *Thysanosoma actinoides*, *Moniezia* spp., *Cysticercus tenuicollis*, *C. tarandi*, and *Echinococcus granulosus* (54). The pathogenicity of the first four species in wapiti is poorly established.

Eggs or proglottids of *E. granulosus* are ingested while wapiti are grazing on areas contaminated by feces from carnivores. These eggs (or proglottids) develop into larvae that cause hydatid disease. Large, thick-walled, fluid-filled hydatid cysts usually develop in the lungs or liver of infected wapiti. Associated clinical signs are minimal except in cases of aberrant migration, or very heavy infections, that reduce the functional capacity of affected organs (79).

#### Mange

Psoroptic mange, or scabies caused by *Psoroptes cervinus*, has been described in wapiti (80–82). Mange in Rocky Mountain wapiti of Jackson Hole, Wyoming, is seen every winter in mature bulls and old cows (80). A recent study of wapiti mortality in Wyoming (82) showed that 63 of 101 of the adult bulls that died had gross evidence of mange, and the highest mortality for this group was during January, the coldest month of the year. The rut (which causes depletion of body energy reserves), injury, and poor winter nutrition, are important factors leading to infestation by *P. cervinus* (80). Clinical signs associated with infestation have included emaciation, paresis, severe alopecia, and dermatitis (81).

#### Ticks

*Dermacentor albipictus*, or winter tick, is found on wapiti and other ungulates of the western plains and Rocky Mountains of Canada and the northwestern United States (54,56,83). Heaviest tick loads have been associated with dense wapiti populations and forage stand depletion (56). Winter tick is a one-host parasite that can cause problems of epizootic proportions in moose and caribou populations (84). In wapiti herds, serious tick infestations have been reported to cause anemia and paresis among calves on winter feeding grounds (56). It would appear that heavy tick loads, combined with other adverse conditions, lead to winter mortality in free-ranging wapiti. Infestation with *D. albipictus* occurs among farmed wapiti but, in this situation, it is more of a cosmetic than life threatening problem (W.M. Samuel, personal communication).

#### Plant toxicoses

Plant poisoning is a major concern to the livestock industry in many areas of the world, but there is little knowledge of its prevalence in wild species. The coevolution of plants and animals may explain the apparent adaptation or tolerance of wild species to doses of toxic plants that would not be tolerated by domestic animals (85).

Both experimental and naturally occurring locoism caused by locoweed (*Astragalus* spp.) have been described in wapiti (86). Clinical signs range from chronic weight loss and depression to incoordination, muscle tremors, and paresis. Affected animals have consistently been emaciated and had serous atrophy of adipose tissue. Lesions of the CNS described in locoweed toxicosis include extensive neuronal swelling and vacuolation with axonal degeneration (86).

## Fungal diseases

Fungal infections are relatively rare in cervids. Mycotic pneumonia and mycotic-induced enteritis have been reported in wapiti (1,87). Mycotic agents have been incriminated in pneumonia, encephalitis, rumenitis, and generalized visceral disease in wapiti in western Canada and the United States (1), and in young farmed red deer in Scotland (87). Pulmonary damage by *Dictyocaulus* spp. is felt to be a potential predisposing factor to the establishment of *Aspergillus* pneumonia (21).

## Conclusion

At this time in Canada and the United States, the major bacterial diseases of wapiti are brucellosis (one focus), clostridial diseases, coliform enteritis of neonates, pasteurellosis, and necrobacillosis. The importance of Johne's disease among wapiti is unknown at this time, but the difficulty in detecting this disease early in its course, and the persistent nature of the infection, makes it a concern. Tuberculosis and brucellosis are of concern because of their zoonotic nature and their potentially devastating effects on both free-ranging and captive ungulates, and the livestock industry.

Of the numerous viral agents that cause health problems in domestic animals, with the exception of coronavirus in neonates, there are few of biological or economic significance in populations of wapiti. This situation could change if new agents not yet reported in North American cervids, such as MCF or HVC-1, became established here.

The potential spread of *Parelaphostrongylus tenuis* has become a high priority issue for those involved with free-ranging or captive ungulates. The increasing movement of captive cervids from east to west in North America increases chances of moving the parasite from enzootic eastern regions to the west where it does not exist. Research is presently underway in Alberta to determine the lethal dose of larvae, whether *P. tenuis* becomes patent in wapiti, and whether *P. tenuis* is always fatal (W.M. Samuel, personal communication). The larvae of *P. tenuis* in feces cannot be distinguished from those of other species in related genera. *Elaphostrongylus cervi* is important because of the largely naive population of North American ungulates, and the infected status of cervid species in some of the exporting countries. There is research presently underway in cervids to determine more about the ecology, species susceptibility, and carrier states associated with *E. cervi* infection (M.W. Lankester, personal communication). For both these parasites, the difficulty in detecting infection increases the concern. *Elaeophora schneideri* causes serious but fairly localized problems for wapiti at the present time. With the interprovincial, international, and farm-to-farm movement of wapiti, fluke control may become a significant concern. There is a pilot study underway in Alberta to determine the efficacy of a benzimidazole drug against *F. magna* in wapiti (M.J. Pybus, personal communication). Lungworm, and possibly liver fluke, are pathogens which threaten the health of wapiti, while gastrointestinal nematodes do not appear to be of great significance.

## Acknowledgments

I would like to thank Drs. Bill Samuel (Department of Zoology, University of Alberta), Ted Leighton (Department of Veterinary Pathology, Western College of Veterinary Medicine), and Jerry Haigh (Department of Herd Medicine and Theriogenology, WCVN) for their patient and helpful input with this review.

CVJ

## References

1. Smits JEG. Elk disease survey in western Canada and north-western United States. Second International Symposium on the Biology of Deer 1990. New York: Springer-Verlag, (in press).
2. Thorne ET, Morton JK, Thomas GM. Brucellosis in elk I. Serologic and bacteriologic survey in Wyoming. J Wildl Dis 1978; 14: 74-81.
3. Thorne ET, Morton JK, Blunt FM, Dawson HA. Brucellosis in elk II. Clinical effects and means of transmission as determined through artificial infections. J Wildl Dis 1978; 14: 280-291.
4. Mulvaney C. Clostridial diseases: Clinical occurrence, control and prevention. Proc Deer Course for Veterinarians, NZ Vet Assoc 1981: 113-116.
5. Buxton D. Alimentary system. In: Alexander TL, ed. Management and Diseases of Deer. 1st ed. London: Veterinary Deer Society, 1986: 77-85.
6. Howe DL. Miscellaneous bacterial diseases. In: Davis JW, Karstad LH, Trainer DO, eds. Infectious Diseases of Wild Mammals. 2nd ed. Ames: Iowa State University Press, 1981: 418-422.
7. Wilson PR. Vaccination of deer: Clostridial diseases. Proc Deer Course for Veterinarians, NZ Vet Assoc 1984: 110-116.
8. Robinson RM. Enterobacterial disease. In: Davis JW, Karstad LH, Trainer DO, eds. Infectious Diseases of Wild Mammals. 2nd ed. Ames: Iowa State University Press, 1981: 320-322.
9. Wilson PR. Diseases of farmed deer. In: Deer Refresher Course Proc 72. Sydney, Australia: Post-graduate Committee in Veterinary Science, University of Sydney, 1984: 505-530.
10. Williams ES, Snyder SP, Martin KL. Pathology of spontaneous and experimental infection of North American wild ruminants with *Mycobacterium paratuberculosis*. Vet Pathol 1983; 20: 274-291.
11. Vance HN. Johne's disease in a European red deer. Can Vet J 1961; 2: 305-307.
12. McKelvey WAC. Johne's disease in deer. Reid HW, ed. Publication of the Veterinary Deer Society, 1987; 2: 24-28.
13. Gilmour N, Nyange J. Paratuberculosis (Johne's disease) in deer. In Practice 1989: 193-196.
14. Jessup DA, Abbas B, Behymer D, Gogan P. Paratuberculosis in tule elk in California. J Am Vet Med Assoc 1981; 179: 1252-1254.
15. Gilmour NJL. A new test for the diagnosis of Johne's disease (paratuberculosis). Publication of the Veterinary Deer Society, 1989; 4: 7.
16. Allred WJ, Brown RC, Murie OJ. Disease kills feedground elk. Wyoming Wildl 1944; 9: 1-8.
17. Rosen MN. Necrobacillosis. Pasteurellosis. In: Davis JW, Karstad LH, Trainer DO, eds. Infectious Diseases of Wild Mammals. 2nd ed. Ames: Iowa State University Press, 1981: 244-252, 332-338.
18. Franson JC, Smith BL. Septicemic pasteurellosis in elk (*Cervus elaphus*) on the United States National Elk Refuge, Wyoming. J Wildl Dis 1988; 24: 715-717.
19. Stumpff CD. Epidemiological study of the outbreak of bovine TB in confined elk herds. Proc US Anim Health Assoc 1982; 86: 524-527.
20. Kollias GV, Thoen CO, Fowler ME. Evaluation of comparative cervical tuberculin skin testing in cervids naturally exposed to mycobacteria. J Am Vet Med Assoc 1982; 181: 1257-1262.
21. Munro R. Respiratory system. Tuberculosis. In: Alexander TL, ed. Management and Diseases of Deer. 1st ed. London: Veterinary Deer Society, 1986: 65-76, 157-160.
22. Beatson NS, Hutton JB. Tuberculosis in farmed deer. Proc Deer Seminar for Veterinarians. NZ Vet Assoc 1981: 143-151.

23. Toma S. Human and nonhuman infections caused by *Yersinia pseudotuberculosis* in Canada from 1962 to 1985. *J Clin Microbiol* 1986; 465-466.
24. Mackintosh CG, Henderson TG. Survey of red deer stags for yersiniosis at slaughter. In: Fennessey PF, Drew KR, eds. *Biology of Deer Production*. Bulletin 22. Wellington, NZ: Royal Society of New Zealand, 1985: 159-162.
25. Mackintosh CG, Henderson TG. The epidemiology of yersiniosis in deer. *Proc Deer Branch. NZ Vet Assoc* 1984; 1: 34-42.
26. Langford EV. *Yersinia enterocolitica* isolated from animals in the Fraser Valley of British Columbia. *Can Vet J* 1972; 13: 109-113.
27. Martyny JW, Botzler RG. *Yersiniae* isolated from wapiti. *J Wildl Dis* 1976; 12: 386-389.
28. Blake JB, McLean BD, Gunn A. Yersiniosis in free-ranging muskoxen on Banks Island, N.W.T., Canada. *Proc 2nd International Muskox Symposium*, Saskatoon, Saskatchewan, Canada, 1-4 Oct, 1987. Flood P, ed. Ottawa: National Research Council of Canada, 1989: A58.
29. Kuttler KL. *Anaplasma* infections in wild and domestic ruminants: A review. *J Wildl Dis* 1984; 20: 12-20.
30. Howe DL, Hepworth WG, Blunt FM, Thomas GM. Anaplasmosis in big game animals: Experimental infection and evaluation of serological tests. *Am J Vet Res* 1964; 25: 1271-1275.
31. Martyny JW, Botzler RG. *Listeria monocytogenes* isolated from wapiti (*Cervus elaphus roosevelti*). *J Wildl Dis* 1975; 11: 330-334.
32. Brown ME. Reproductive disorders-female. In: Alexander TL, ed. *Management and Diseases of Deer*. 1st ed. London: Veterinary Deer Society, 1986: 112.
33. Kingscote BF, Yates WDG, Tiffin GB. Diseases of wapiti utilizing cattle range in southwestern Alberta. *J Wildl Dis* 1987; 23: 86-91.
34. Adrian WJ, Keiss RE. Survey of Colorado's wild ruminants for serological titres to brucellosis and leptospirosis. *J Wildl Dis* 1977; 13: 429-431.
35. Dulac GC, Dubuc C, Myers DJ, Afshar A, Taylor EA. Incursion of bluetongue virus type II and epizootic hemorrhagic disease of deer type 2 for two consecutive years in the Okanagan Valley. *Can Vet J* 1989; 30: 351.
36. Murray JO, Trainer DO. Bluetongue virus in North American elk. *J Wildl Dis* 1970; 6: 144-148.
37. Hoff GL, Trainer DO. Experimental infection in North American elk with epizootic hemorrhagic disease virus. *J Wildl Dis* 1973; 9: 129-132.
38. Howell NA, Kumm NA, Botha MJ. The application of improved techniques to the identification of strains of bluetongue virus. *Onderstepoort J Vet Res* 1970; 37: 59-66.
39. Karstad LH. Miscellaneous viral infections. In: Davis JW, Karstad LH, Trainer DO, eds. *Infectious Diseases of Wild Mammals*. 2nd ed. Ames: Iowa State University Press, 1981: 202-211.
40. Horner GW, Read DH. Parapox infection in deer: A new disease? *Proc Deer Course for Veterinarians. Deer Branch, NZ Vet Assoc* 1986: 132-137.
41. Williams ES, Young S. Spongiform encephalopathy of Rocky Mountain Elk. *J Wildl Dis* 1982; 18: 465-471.
42. Kimberlin RH. Transmissible encephalopathies in animals. *Can J Vet Res* 1990; 54: 30-37.
43. Nettleton JA, Sinclair JA, Herring DM, et al. Prevalence of herpesvirus infection in British red deer and investigations of further disease outbreaks. *Vet Rec* 1986; 118: 267-270.
44. Thiry E, Vercouter M, Dubuisson J, et al. Serological survey of herpesvirus infections in wild ruminants of France and Belgium. *J Wildl Dis* 1988; 2: 268-273.
45. McAllum HJF, Mavor NM, Hemmingsen P. A malignant catarrhal fever-like disease in red deer (*Cervus elaphus*) in New Zealand. *NZ Vet J* 1982; 30: 99-101.
46. Beatson NS. Malignant catarrhal fever — field progress. *Proc Deer Course for Veterinarians. Deer Branch, NZ Vet Assoc* 1984; 1: 117-121.
47. Beatson NS. Field observations of malignant catarrhal fever in red deer in New Zealand. In: Fennessey PF, Drew KR, eds. *Biology of Deer Production*. Bulletin 22. The Royal Society of New Zealand, 1985: 135-137.
48. Orr MB, Mackintosh CG. An outbreak of malignant catarrhal fever in Pere David's deer (*Elaphurus davidianus*). *NZ Vet J* 1988; 1: 19-21.
49. Wobeser G, Majka JA, Mills JHL. A disease resembling malignant catarrhal fever in captive white-tailed deer in Saskatchewan. *Can Vet J* 1973; 14: 106-109.
50. Webb PA, McLean RG, Smith GC, et al. Epizootic vesicular stomatitis in Colorado, 1982: Some observations on the possible role of wildlife populations in an enzootic maintenance cycle. *J Wildl Dis* 1987; 23: 192-198.
51. Schneider LG, Muller WW, Hohnsbeen KP. Rabies in Europe 1st quarter 1989. *Rabies Bulletin Europe*. 1989; 13: 1-9, 15-28.
52. Reid HW. Rabies. In: Alexander TL, ed. *Management and Diseases of Deer*. 1st ed. London: Veterinary Deer Society, 1986: 146.
53. Leighton FA, Williams ES. Intracytoplasmic neuronal inclusions in the hippocampus of non-rabid moose, *Alces alces*. *J Wildl Dis* 1983; 19: 285-288.
54. Worley DE. Parasites and parasitic diseases of elk in the Northern Rocky Mountain region: A review. In: Boyce MS, Haydon-Wing LD, eds. *North American Elk: Ecology, Behavior and Management*. University of Wyoming, 1980: 206-211.
55. Worley DE, Barrett RE, Presidente, PJA, Jacobson RH. The Rocky Mountain elk as a reservoir host for parasites of domestic animals in western Montana. *Bull Wildl Dis Assoc* 1969; 5: 348-350.
56. Flook DR, Stenson JE. Incidence and abundance of certain parasites in wapiti in the national parks of the Canadian Rockies. *Can J Zool* 1969; 47: 795-803.
57. Watson TG, Charleston WAG. The significance of parasites in farmed deer. *Biology of Deer Production*. Bulletin 22. The Royal Society of New Zealand, 1985: 105-117.
58. Throlson K. Bison health. In: *Bison Breeders' Handbook*, 2nd ed. Denver: American Bison Association, 1988: 40-59.
59. Bergstrom RC. Prevalence of *Dictyocaulus viviparus* infection in Rocky Mountain Elk in Teton County, Wyoming. *J Wildl Dis* 1975; 11: 40-44.
60. Barrett RE, Worley DE. The incidence of *Dictyocaulus* sp. in three populations of elk in south-central Montana. *Bull Wildl Dis Assoc* 1966; 2: 5-6.
61. Presidente PJA, Worley DE, Catlin JE. Cross-transmission experiments with *Dictyocaulus viviparus* isolates from Rocky Mountain elk and cattle. *J Wildl Dis* 1972; 8: 57-62.
62. Corrigan W. *Dictyocaulus viviparus* infections in red deer. In: Fennessey PF, Drew KR, eds. *Biology of Deer Production*. Bulletin 22. The Royal Society of New Zealand, 1985: 123-126.
63. Carpenter JW, Jordan HE, Ward BC. Neurological disease in wapiti naturally infected with meningeal worms. *J Wildl Dis* 1973; 9: 148-153.
64. Anderson RC, Prestwood AK. Lungworms. In: Davidson WR, Hayes FA, Nettles VF, Kellogg FE, eds. *Diseases and Parasites of White-tailed Deer*, 1981: 266-317.
65. Olsen A, Woolf A. A summary of the prevalence of *Parelaphostrongylus tenuis* in a captive wapiti population. *J Wildl Dis* 1979; 15: 33-35.
66. Pybus MJ, Samuel WM, Crichton V. Identification of dorsalspined larvae from free-ranging wapiti (*Cervus elaphus*) in southwestern Manitoba, Canada. *J Wildl Dis* 1989; 25: 291-293.
67. Bindernagel JA, Anderson RC. Distribution of the meningeal worm in white-tailed deer in Canada. *J Wildl Manage* 1972; 36: 1349-1353.
68. Anderson RC, Lankester MW, Strelive UR. Further experimental studies of *Pneumostrongylus tenuis* in cervids. *Can J Zool* 1966; 44: 851-861.
69. Karns PD. *Pneumostrongylus tenuis* from elk (*Cervus canadensis*) in Minnesota. *Bull Wildl Dis Assoc* 1966; 2: 79.
70. Lankester MW, Northcott TH. *Elaphostrongylus cervi* Cameron 1931 (Nematoda: Metastrongyloidea) in caribou (*Rangifer tarandus caribou*) of Newfoundland. *Can J Zool* 1979; 57: 1384-1392.
71. Mason PC, McAllum HJF. *Dictyocaulus viviparus* and *Elaphostrongylus cervi* in wapiti in New Zealand. *NZ Vet J* 1976; 24: 23.
72. English AW, Watt CF, Corrigan W. Larvae of *Elaphostrongylus cervi* in the deer of Scotland. *Vet Rec* 1985: 254-256.
73. Sugar L, Kavai A. On the occurrence of *Elaphostrongylus cervi* Cameron, 1931, in a red deer population in Hungary. *Parasit Hung* 1977; 10: 95-96.

74. Hibler CP, Adcock JL, Davis RW, Abdelbaki YZ. Elaeophorosis in deer and elk in the Gila Forest, New Mexico. *Bull Wildl Dis Assoc* 1969; 5: 27-30.
75. Hibler CP, Adcock JL. Elaeophorosis. In: Davis JW, Anderson RC, eds. *Parasitic Diseases of Wild Mammals*. Ames: Iowa State University Press, 1971: 263-278.
76. Wobeser GA, Gajadhar AA, Hunt HM. *Fascioloides magna*: Occurrence in Saskatchewan and distribution in Canada. *Can Vet J* 1985; 26: 241-244.
77. Pybus M. A parasite survey of big game in Alberta. *J Wildl Dis* 1990; 26: (in press).
78. Dunn AM. *Fascioloides magna* infection. In: *Veterinary Helminthology*, 2nd ed. London: Heinemann Medical, 1978: 201.
79. Leiby PD, Dyer WG. Cyclophyllidean tapeworms of wild carnivora. In: Davis JW, Anderson RC, eds. *Parasitic Diseases of Wild Mammals*. Ames: Iowa State University Press, 1971: 174-234.
80. Murie OJ. *The Elk of North America*. Harrisburg, Pennsylvania: The Stackpole Co., 1951.
81. Colwell DA, Dunlap JS. Psoroptic mange in a wapiti. *J Wildl Dis* 1975; 11: 66-67.
82. Smith BL. Scabies and elk mortality on the national refuge, Wyoming. *Proc 1984 Western States and Provinces Elk Workshop*. Alberta Fish and Wildl Div, 1984: 180-195.
83. Stelfox JG. Liver, lung and larvae/parasites and diseases in moose, deer and wapiti in Alberta. *Land-Forest-Wildl* 1962; 5: 5-12.
84. Wobeser GA. Ectoparasites. In: *Handbook of Diseases of Saskatchewan Wildlife, Saskatchewan Parks and Renewable Resources*, 1985: 41-43.
85. Fowler ME. Plant poisoning in free-living wild animals: a review. *J Wildl Dis* 1983; 19: 34-43.
86. Adcock JL, Keiss RE. Locoism in wapiti. *Bull Wildl Dis Assoc* 1969; 5: 121-124.
87. Munro R, Hunter AR, Boniwell M, Corrigan W. Systemic mycosis in Scottish red deer (*Cervus elaphus*). *J Comp Pathol* 1984; 95: 281-289.

## PRACTITIONERS' CORNER

## LE COIN DES PRATICIENS

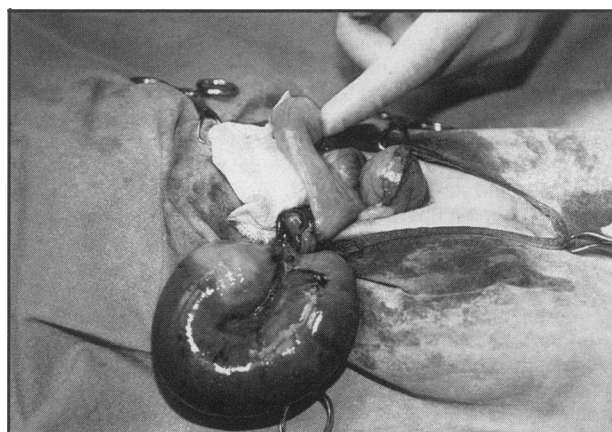
### Uterine torsion in a cat

James D. Young, George P. Hillis, Marie L. McKibbin

A three-year-old female domestic short-haired cat was presented for post-partum evaluation. This was her third litter and both previous queenings were uneventful. The cat had queened one kitten 24 hours previously without any difficulty and had not demonstrated any further signs of labor. The kitten was reported to be nursing well and was apparently healthy. The queen's appetite was good and she was urinating and defecating normally, with no vaginal discharge, and was in no apparent discomfort. It was unknown whether the placenta was passed with the kitten. A slight abdominal distension had been present for at least two weeks on the right side.

On physical examination the cat was quiet, alert and responsive. The heart rate, respiratory rate, mucous membrane color, capillary refill time, and temperature were all within normal ranges. No vaginal discharge was evident. Abdominal palpation revealed a 6 × 3 × 3 cm firm, non-painful mass within the cranial right paralumbar fossa. The mass was freely moveable and of a doughy consistency.

Lateral and ventrodorsal radiographs revealed two small fetuses in the right cranial quadrant of the abdomen. No other abnormalities were noted. A mid-line laparotomy was performed to remove the fetuses. The uterus was exteriorized (Figure 1) and revealed a 360° torsion of the right uterine horn; the left horn appeared to be normal. The twisted uterine horn was dark purple, edematous and moderately friable. An ovariohysterectomy was performed and recovery was uneventful. The two fetuses were macerated and were



**Figure 1.** Torsion of the right uterine horn in a three-year-old cat.

approximately one half the size of a term kitten. It was estimated that the torsion probably occurred between day 40 and day 50 of gestation.

Uterine torsion in the cat is considered to be a rare disease and usually occurs in late pregnancy. This case was unusual because of the absence of abdominal pain, vaginal discharge and signs of shock. Once uterine torsion is suspected, an immediate exploratory laparotomy must be carried out. The cause of uterine torsion is not known.

#### References

1. Morrow DA. *Current Therapy in Theriogenology*. 2nd ed. Toronto: WB Saunders, 1986: 814-815.
2. Ettinger SJ. *Textbook of Veterinary Internal Medicine*. 3rd ed. Toronto: WB Saunders, 1989: 1801.

East Oshawa Animal Hospital, 1 Townline Road North, Oshawa, Ontario L1K 1A7